

# Chapter 1

## Diseases of the Oral Cavity and Esophagus

Timothy H. Ogilvie

By: Dr.Stator = thanatomania 2008  
Egyptian Muslim veterinarian  
dr\_stator\_veto\_18486@yahoo.com

**I. STOMATITIS** is inflammation of the oral cavity. Conditions include glossitis (inflammation of the lingual mucosa), palatitis or **lampas** (inflammation of the palate), and gingivitis (inflammation of the mucosa and gums).

**A.** Patient profile and history. The patient profile is variable. Domestic animals of any breed, sex, or age may be affected by a painful mouth for a variety of reasons. The history is also highly variable and is often nonspecific for any particular condition.

**B.** Clinical findings. Inflammatory lesions of the mouth are clinically characterized by partial or complete loss of appetite, painful or slow mastication, and prehension difficulties. Sialism or ptyalism, fetid breath (if necrotic tissue is present), and local lymphadenopathy (if there has been bacterial invasion of tissue) may also be present.

**C.** Etiology and **pathophysiology**. Stomatitis may be caused by physical, chemical, or infectious agents.

1. Physical agents. Foreign objects (e.g., corn cobs, sticks, rocks) may become lodged in the lingual groove of cattle or in the upper dental arcade of horses. Injury may also be inflicted by equipment used to administer oral medications (e.g., balling guns, dosing syringes) or by dental instruments (e.g., floats, files).
2. Chemical agents. Irritant substances (e.g., mercuric preparations used as counterirritants) may be inadvertently licked or eaten by an animal.
3. Infectious agents. Stomatitis may be caused by bacteria, fungi, or **viruses** (Table 1-1).

**D.** **Diagnostic** plan. A complete physical examination and history is often necessary to determine the location of the problem and define its cause. It must be determined whether the clinical signs are caused by local lesions, or whether they are manifestations of systemic disease.

**E.** **Laboratory** tests are usually not performed for conditions that seem to be localized to the mouth.

- t. Hematologic work-up. A hematologic work-up is valuable if systemic involvement is suspected.
2. Bacterial culture may be helpful. For example, crushing and gram-staining the sulfur granules from the exudate of an actinobacillosis lesion will reveal *Actinobacillus lignieresii*.

**F.** Differential diagnoses include:

1. Neurologic diseases (e.g., rabies, equine leukoencephalomalacia), which may cause excessive salivation
2. **Sialadenitis**
3. Toxicities (e.g., organophosphatotoxicity)
4. **Actinomycosis** (lumpy jaw), an osteomyelitis of the mandible or maxilla (see Chapter 42)

**G.** Therapeutic plan

1. Physical injury. In general, lesions of the mouth and tongue that have been produced

TABLE 1-1. Infectious Causes of Stomatitis

Causative Agent	Clinical Disease
<i>Fusobacterium necrophorum</i> (B)	Oral necrobacillosis (necrotic stomatitis)
<i>Actinobacillus lignieresii</i> (B)	Actinobacillosis (wooden tongue)
<i>Candida albicans</i> (F)	Stomatitis, glossitis
Aphthovirus (V)	Foot and mouth disease
Enterovirus (V)	Swine vesicular disease
Vesicular stomatitis virus (V)	Vesicular stomatitis
Bovine viral diarrhea virus (V)	Bovine viral diarrhea, mucosal disease
Bovine malignant catarrh virus (V)	Bovine malignant catarrh
Rinderpest virus (V)	Rinderpest
Bovine papular stomatitis virus (V)	Bovine papular stomatitis
Contagious ecthyma virus (V)	Contagious ecthyma
Vesicular exanthema virus (V)	Vesicular exanthema of swine
Bluetongue virus (V)	Bluetongue
Epizootic hemorrhagic disease virus (V)	Epizootic hemorrhagic disease (deer)

B = bacteria; F = fungus; V = virus.

by physical agents heal rapidly without interventional therapy. Supportive care may be offered in the form of free-choice water and a soft, palatable diet for a few days.

- Chemical injury. The oral cavity should be flushed with water immediately, and the animal should be observed closely to determine if any ingestion has occurred. Free-choice water and a soft, palatable ration should be offered. If anorexia (as a result of the oral cavity lesions) occurs, intravenous fluid therapy and an indwelling nasogastric tube may be necessary to meet fluid and nutritional demands until healing occurs. With young or small patients, total parenteral nutrition may be a viable and economic option.
- Infection.** Specific therapies are outlined in 11.

**H.** Prevention. Many cases of stomatitis can be prevented by providing the client with information regarding management, feeding care, and hygiene.

#### **I.** Specific condition

##### 1. Necrotic stomatitis (oral **necrobacillosis**)

- Patient profile and history. Necrotic stomatitis occurs in young, milk-fed calves. The condition is often linked to unsanitary management and unhygienic feeding utensils.
- Clinical findings. Necrotic stomatitis is characterized by fever, depression, anorexia, pyalism, and painful swallowing. In classic oral necrobacillosis, the lesions involve the buccal mucosa, giving the animal a **puffy-cheeked** appearance. A characteristic foul smell is associated with the exudate in the mouth.
- Therapeutic plan. Oral necrobacillosis usually responds well to penicillin G procaine (**10–20,000 IU/kg** intramuscularly, twice daily). Treatment may be required for **7–10** days.

##### 2. Actinobacillosis (wooden **tongue**) is usually a sporadic condition. In cattle, the tongue, and less commonly, the pharyngeal lymph nodes, are involved, whereas in sheep, the soft tissues of the mouth, face, and neck are affected.

- Clinical findings. Onset is acute and characterized by pyalism, excessive tongue movement, and an inability to eat
  - In cattle, the tongue is swollen, hard, and painful on manipulation. The tongue may be enlarged early in the course of the disease, but later it becomes shrunken and firm.
  - Nodules and ulcers may be present. Suppuration occurs and pus is discharged from the affected areas.

(3) Starvation may eventually occur because of pain or the inability to prehend and masticate food.

- Etiology and pathophysiology. *Actinobacillus lignieresii*, a gram-negative rod, is a normal inhabitant of the mouth of many ruminants. The organism is thought to gain entrance to the soft tissues of the oral cavity through abrasions and penetrating wounds of the mouth and tongue, leading to the development of a **granulomatous** abscessation.
- Differential diagnoses for this condition include penetrating foreign bodies, bacterial phlegmon, abscesses, and tuberculosis involving the lymph nodes of the head and neck.
- Therapeutic plan. It is important to treat the condition quickly, control discharges, and isolate affected animals. The standard treatment is iodides in combination with antibiotics.
  - Potassium iodide is administered orally (**6–10 g/day** for **1–10** days). Sodium chloride is administered intravenously (**1 g/12 kg** as a 10% solution). Treatment with sodium chloride may be repeated once.
  - Signs of iodism (e.g., lacrimation, anorexia, coughing, flaky skin) will often develop with a course of potassium iodide therapy, necessitating adjustment of the dose.

##### 3. Mycotic stomatitis is similar to the human disease, thrush.

- Patient profile and history. Stomatitis caused by fungi or yeasts is most commonly seen in hand-reared young (lambs, piglets) or as a **sequela** to long-term oral antibiotic therapy. The condition is often associated with unhygienic feeding utensils.
- Clinical findings. The lesions appear as white spots or as a velvety white membrane covering the mucosa. The white plaques progress to shallow ulcers.
- Therapeutic plan. Animals may be treated with mild oral antiseptic solutions (e.g., **2%** copper sulfate). If overuse of oral antibiotics is the cause of the stomatitis, the antibiotic should be discontinued and the rationale for its initial use investigated.

##### 4. Vesicular stomatitis

- Patient profile and history. Vesicular stomatitis occurs in cattle, horses, swine, and, occasionally, humans. The disease is most common in adult cattle.
- Clinical findings. Vesicular stomatitis is usually sporadic in occurrence, but up to **60%** of a herd may develop clinical signs. The disease often develops in the summer or fall and may be cyclical in occurrence, with many years elapsing between outbreaks.
  - Cattle exhibit fever, excessive salivation, and anorexia resulting in weight loss. By the time the disease is recognized, vesicles may have been replaced by erosions and ulcers on the lips, gums, dental pad, and tongue. Teat lesions, which cause **mastitis** and a drop in milk production, are also found; coronary band lesions and lameness are less common. Recovery generally occurs within **2–21** days; the lesions resolve after **1–2** months.
  - Horses exhibit a transient pyrexia with vesicles on the lips and tongue. Hyperemia and ulceration of the coronary band area is common. Lesions may also involve the nasal turbinates and nasopharynx, causing epistaxis.
  - Swine. Vesicles and ulcers are found on the snout, oral mucosa, and feet.
  - Humans. Fever and influenza-like symptoms have been described.
- Etiology and pathogenesis
  - Etiology. Vesicular stomatitis is caused by vesicular stomatitis virus (**VSV**), a **rhabdovirus**. The New Jersey and Indiana strains are the main **serotypes**.
  - Pathogenesis. The pathogenesis of vesicular stomatitis is not fully understood, but it is hypothesized that VSV is a plant virus that undergoes modification once it is ingested by insects that feed on the plants.
    - Transmission. Insects may spread the modified virus to other plants or directly to animals. Animals may also become infected through consumption of infected plants, by direct transmission, through human transmission, or via fomites (e.g., milking machines).

TABLE 1-2. Viral Vesicular Disease Infection Profile

Disease	Cattle	Sheep and Goats	Pigs	Horses	Humans
Vesicular stomatitis	+++	+	+++	+++	+
Foot and mouth disease	+++	+++	+++	+	+
Vesicular exanthema	-	-	+++	-	+
Swine vesicular disease	-	-	+++	-	+

- = does not occur in the species in a controlled exposure; + = sometimes occurs in the species in a controlled exposure; +++ = often occurs in the species in a controlled exposure; ++++ = frequently occurs in the species in a controlled exposure.

- (i) The seasonal disappearance of the disease in temperate regions may correspond to a decrease in the insect population or elimination of fresh forages from the diet.
    - (ii) The disease is **enzootic** in areas of warm temperature, high rainfall, and heavy insect populations (e.g., the tropics or subtropics).
  - (b) Portal of entry. It is thought that mucous membrane abrasions are necessary for virus penetration.
  - d. Diagnostic plan. Because the ulcers in this condition and the disease pattern closely resemble those of foot and mouth disease (see 115; Table 1-2), federal authorities must be notified and a herd quarantine must be imposed until the diagnosis is confirmed. Virus isolation, although difficult, is attempted from fresh vesicular fluids and from epithelial biopsies. Serum complement fixation and fluorescent antibody tests are used to evaluate serum.
  - e. Therapeutic plan. Vesicular stomatitis will usually die out within the herd, but affected animals should be isolated from herd mates. Free-choice water and good quality feed should be provided, and soothing, **lanolin-based** teat dips should be used on cows with teat lesions.
  - f. Prevention. Insect control should be employed to prevent the spread of the disease. Affected animals should be handled, fed, and milked last.
5. Foot and mouth disease
- a. Patient profile and history. Cattle and swine of any age and breed are most susceptible. Foot and mouth disease primarily affects wild and domestic **cloven-hooved** animals in Africa, Europe, Asia, and South America. Outbreaks have occurred in previously disease-free areas (e.g., North America) as a result of movement of animals or meat products, but the outbreaks have always been successfully controlled.
  - b. Clinical findings
    - (1) **Cattle.** In cattle, this is a **multisystemic** disease characterized by fever and depression followed by stomatitis, lameness, and mastitis. Secondary bacterial infection of the lesions occurs as the thin-walled vesicles rapidly progress to ulcers.
      - (a) Oral lesions. Vesicles and **bullae** appear on the tongue, buccal **mucosa**, dental pad, and gingiva, causing excessive salivation and painful mastication.
      - (b) Foot lesions. Vesicles appear on the feet, between the claws, and on the coronary band, producing lameness.
      - (c) Teat lesions. Vesicular lesions develop on the teats, resulting in pain and an unwillingness of the animal to be milked.
    - (2) Swine, sheep, and goats. In these animals, the presentation is similar to that in cattle, but the clinical signs are usually milder.
  - c. Etiology and pathogenesis. Foot and mouth disease is caused by a picornavirus of the genus **Aphthovirus**. The virus is resistant and may persist on fomites or in meat products; it is also readily transmitted by carrier animals (e.g., swine, sheep, goats). In the latter case, the mode of transmission is via inhalation or ingestion.
  - d. **Diagnostic** plan. Federal authorities must be notified. Virus identification is

accomplished using antibody detection tests (e.g., complement fixation, virus neutralization) or antigen identification assays [e.g., enzyme-linked immunosorbent assay (ELISA)]. In foot and mouth virus-free areas, a quarantine, test, and slaughter policy is usually evoked in response to identification of the disease.

- e. Prevention. Mutations occur constantly, making a consistent vaccination program impractical. Because economic losses (as a result of production inefficiencies and trade restrictions) can be substantial, type-specific vaccines for the region are often employed in endemic areas.
6. Swine vesicular disease
- a. Patient profile and history. This disease only affects pigs and is foreign to North America. It is significant because of its clinical similarity to foot and mouth disease.
  - b. Clinical findings. The vesicles found with this disease are most prominent around the coronary band and between the claws of the feet and less prominent in the mouth. Unlike the vesicles of foot and mouth disease, the vesicles of swine vesicular disease are thick-walled; therefore, they are not easily ruptured and may persist for 1–2 days. A transient fever accompanied by anorexia is often seen. Animals may be mildly to moderately lame.
  - c. Etiology and pathogenesis. Swine vesicular disease is caused by an enterovirus. Because the virus can survive and persist on fomites and in meat products, control through disinfection and hygiene is difficult. Infection occurs via oral abrasions and the morbidity rate is 100%.
  - d. Diagnostic plan. Federal authorities should be notified because of the similarity of swine vesicular disease to foot and mouth disease.
  - e. Therapeutic plan. Treatment is not warranted for this mild disease and is rarely attempted.
7. Vesicular exanthema
- a. Patient profile and history. Only feral and domestic swine are affected, although experimental transmission has been successful in horses. The disease is limited to the Southwestern United States.
  - b. Clinical findings include a high fever, depression, anorexia, and vesicular lesions in the mouth, on the snout, teats, udder coronary band, and heels, and between the claws. The vesicles rupture to ulcers within 24–48 hours, resulting in lameness and secondary infection. Abortion may also occur. Infection may be subclinical.
  - c. Etiology and pathogenesis. The disease is caused by a calicivirus harbored by sea lions. The virus is thought to be periodically transmitted to the feral pig population in coastal areas (e.g., California) and then on to domestic pigs. Sources of infection are live pigs and pork products.
  - d. Diagnostic plan. Federal authorities should be notified to rule out other vesicular diseases.
  - e. Therapeutic plan. Treatment is rarely attempted.
8. Bluetongue
- a. Patient profile and history. Bluetongue is most common and virulent in Africa but causes disease in ruminants throughout the world. Bluetongue occurs in many locations in the United States and in parts of western Canada. It affects sheep and, occasionally, cattle.
  - b. Clinical findings
    - (1) **Sheep**
      - (a) Epidemic (acute) disease. Fever and a bloody or mucopurulent nasal discharge are common. Swelling and edema of the mouth, lips, and tongue develop, resulting in dyspnea, dysphagia, and cyanosis of the oral membranes, followed by the development of necrotic oral ulcers. Coronitis and laminitis may lead to lameness and recumbency. Diarrhea, conjunctivitis, lacrimation, and pneumonia may also be seen. In utero infection may result in abortions or the birth of immune-tolerant lambs with or without signs of musculoskeletal or neurologic disorders (e.g., arthrogryposis, hydranencephaly).

- (b) Endemic disease. In endemic areas, sheep may have an increased incidence of abortions and a decreased level of performance.
- (2) Cattle usually have inapparent infections but may show some or all of the same signs as sheep.
- c. Etiology and pathogenesis
- (1) Etiology. Bluetongue is caused by an orbivirus, bluetongue virus (BTV). There are many serotypes of BTV and strains within serotypes. One closely related virus, epizootic hemorrhagic disease virus (EHDV), causes a peracute and fatal hemorrhagic disease in white-tailed deer and pronghorn antelope that is very similar to bluetongue.
- (2) Transmission. BTV is transmitted by biting flies (*Culicoides* species). Cattle and wild African ruminants are reservoirs. The virus is also found in semen, leading to congenital infection.
- d. Diagnostic plan. Clinical diagnosis can be difficult because of the variety of clinical signs and presentations. Outbreaks should be reported to federal authorities. Virus identification can be accomplished through:
- (1) Serologic tests (e.g., complement fixation, indirect immunofluorescence, agar gel immunodiffusion, serum neutralization)
- (2) ELISA
- (3) Animal inoculation (e.g., sheep, chick embryo, or rodent)
- (4) Polymerase chain reaction (PCR) assays
- (5) Enzyme-linked oligonucleotide-sorbent assay (ELOSA)
- e. Therapeutic plan. Treatment is nonspecific and often unrewarding for the acutely affected animal. Nursing care is recommended for the mild form of the disease. Congenital infections are not treated.
- f. Prevention. Because the morbidity rate is 50%–75% and the mortality rate is 20%–50%, production losses can be of major significance; therefore, prevention of the disease is important. A vaccine is available in the United States.
9. Contagious **ecthyma** (contagious pustular dermatitis, **orf**)
- a. Patient profile. Contagious ecthyma is a common disease of sheep and goats.
- Young animals are most commonly affected, but lesions can occur on or around the udders of older animals that lack immunity, presumably following inoculation by nursing lambs.
- b. Clinical findings
- (1) Crusty, proliferative lesions are most commonly found on the mouth, muzzle, and nostrils. The lesions begin as papules that rapidly progress to vesicles, pustules, and, finally, scabs covering a granulated, inflamed base. The lesions are sore and may fissure, crumble, and bleed.
- (2) Animals recover from the condition in approximately 3 weeks, but not before suffering ill-thrift due to decreased feed consumption and a reluctance to nurse. The severity of the disease varies, depending on the location of the lesions (e.g., lesions may extend into the alimentary or respiratory tract). Generally, morbidity is high but mortality is low, unless secondary infections occur or there is extensive spread of the lesions.
- c. Etiology and pathogenesis. Contagious ecthyma is a highly infectious disease caused by a parapoxvirus and spread by direct contact. It can be zoonotic; caretakers may develop scabs on their hands.
- d. Diagnostic plan. The diagnosis is based on the finding of typical clinical signs in the appropriate age-range population.
- e. Differential diagnoses include sheep pox, goat pox, bluetongue, and ulcerative dermatosis.
- f. Therapeutic plan. Contagious ecthyma is usually a self-limiting disease, but nursing care, supportive feeding, or antibiotics (to resolve secondary bacterial infections) may be needed by some patients.
- g. Prevention
- (1) Affected animals should be isolated from the herd or flock if possible.
- (2) Measures to limit transmission (e.g., using separate feeding utensils) should be employed.

- (3) Vaccination programs are common in large sheep-rearing areas. Commercial vaccines are available or autogenous vaccines can be prepared.

10. Bovine papular stomatitis (BPS)
- a. Patient profile. BPS is a common disease in calves or young cattle. It has a worldwide distribution.
- b. Clinical findings
- (1) The early sign of a transient fever usually goes unnoticed.
- (2) The initial lesions are papules that rapidly coalesce and progress to round lesions with a necrotic, grey, depressed center and a raised, reddened periphery. Lesions are found on the muzzle, the hard palate, or inside the nostrils, but not on the tongue.
- (a) The lesions may be found incidentally while examining the mouths of young cattle, or they may be associated with a period of weight loss and diarrhea ("rat-tail" syndrome, which affects feeder cattle).
- (b) The lesions may persist for weeks.
- c. Etiology. BPS is caused by **parapoxvirus**.
- d. Diagnostic plan. The diagnosis is based on the characteristic lesions and the usual lack of any other clinical findings in young cattle. It is important that this benign disease not be confused with other, more serious, diseases that produce oral ulcers or erosions (e.g., vesicular stomatitis).
- e. Therapeutic plan. Therapeutic intervention is unnecessary. Good hygiene should be practiced to avoid secondary infections.

## II. DENTAL CONDITIONS

- A. **Observable** changes in the teeth of ruminants. A variety of inherited, congenital, and developmental conditions can affect the teeth of ruminants. Generally, therapy is only undertaken in valuable animals when the extraction of one or two affected teeth and supplemental feeding might prolong the animal's productive life.

1. Staining. Porphyrinuria stains the teeth of cattle reddish brown.
2. Defective enamel formation. Osteogenesis **imperfecta** causes defective enamel formation.
3. Mottling. Fluoride toxicity (fluorosis) damages the teeth prior to tooth eruption. The teeth appear eroded, mottled, and wear excessively.
4. Excessive wear. Mature animals of any species may be affected as a result of abnormally abrasive diets or rations deficient in calcium. The worn teeth prohibit proper prehension and mastication, resulting in weight loss (or diminished weight gain) and necessitating early **cullage**.
5. **Malpositioning** and excessive rotation of the cheek teeth may occur in goats secondary to osteodystrophia fibrosa.
6. Broken teeth. Trauma most commonly involves the incisor teeth.

### B. Dental conditions of sheep

1. **Dentigerous** cysts, a developmental abnormality, occur most commonly in sheep. The incisors are usually encysted within the alveolar bone of the central mandible.
2. Periodontal disease (periodontitis, alveolar **periostitis**)
  - a. Clinical findings include premature wear and breakage of teeth (broken mouth), painful mastication, and poor feed conversion or weight loss. Gingivitis is the primary lesion.
  - b. Etiology and pathogenesis. A chronic bacterial infection (perhaps caused by *Bacteroides gingivalis*) leads to destruction of the periodontal membrane with resultant alveolitis, osteomyelitis, and bony lysis. The cause of the periodontitis is not

- well defined; it may be multifactorial and related to either the accumulation of debris in the gingival sulcus or a mineral deficiency.
- c. Therapeutic plan. Antibiotics may be used to treat periodontal disease.

### C. Dental conditions of horses

#### 1. Clinical findings

- Feed refusal or selectivity in eating. Grain is often preferred over hay, which has to be masticated more fully.
- Pain. Chewing is a slow process and the animal may toss his head in frustration. Food may be wadded in the mouth or expelled in masses (quidding), or grain may fall out of the mouth while the horse is eating. The animal may hold his head with the affected side of the jaw up. Performance animals may resist the bit or place unilateral tension on a line or rein.
- Weight loss may result from decreased feed intake or decreased digestibility of incompletely masticated feedstuffs. Whole-grain or long-stemmed fiber may be seen in the feces.
- Recurrent esophageal choke or intestinal colic may result from poor digestibility of the food or because water intake is decreased secondary to increased tooth sensitivity.

#### 2. Diagnostic plan

- The animal should be observed while eating, drinking, and working. In all cases, a thorough oral examination is necessary to identify the affected teeth and secondary mouth lesions. The animal should be sedated, and anesthesia may be necessary.
- In some cases, radiographs may be necessary.

#### 3. Specific conditions

- Ectopic teeth are incisors or cheek teeth that grow in an abnormal direction. They are thought to arise from injuries to the tooth bud and should be removed if they are causing problems for the animal.
- Supernumerary teeth are usually extra incisors; rarely, extra cheek teeth are seen. Any number of extra teeth, including whole rows, may be present. Supernumerary teeth are usually left alone unless they are causing problems.
- Wolf teeth are small, singular, vestigial premolars in front of the upper cheek teeth that may cause buccal lacerations or interfere with the bit. Occasionally, they are seen on the lower arcade. Because of their short root structure, they are easily extracted in the standing animal.
- Retained deciduous teeth are common.
  - Occasionally, incisors are retained if the permanent teeth erupt behind them. The retained incisor must be extracted.
  - More commonly, deciduous premolars are retained as caps over the erupting permanent tooth. The cap delays the eruption of the permanent tooth and acts as a hinge, trapping debris and causing buccal irritation. Caps can be removed by filing or with dental elevators or forceps.
- Sharp edges (points) develop on the buccal surface of the upper arcade of cheek teeth and the lingual border of the lower arcade. Points are most common in horses 2–5 years of age and occur when the enamel of the teeth wears unevenly as a result of friction between the narrow mandible and the wider maxillary arcade. These sharp edges can cause mucosal lacerations and may be removed by routine filing or floating.
- Infection
  - Etiology and pathogenesis
    - Infection can occur secondary to trauma or tooth fractures that extend into the pulp cavity.
    - Malocclusions, gingivitis, periodontitis, or cementum defects also predispose to infection.
    - A patent infundibulum allows material to enter the root canal, in many cases creating an abscess. An apical abscess in the mandibular bone looks like a normal eruption cyst, but radiographically it has the

appearance of a proliferative osteomyelitis. An abscess of one of the last four cheek teeth in the upper arcade can cause sinusitis.

#### (2) Therapeutic plan

- Medical therapy is employed initially to attempt preservation of the tooth. Systemic penicillin (22,000 IU/kg twice daily by intramuscular injection) coupled with endoscopy and flushing of the maxillary sinus (if sinusitis is present) may be successful. Radiographic follow-up allows evaluation of the therapy.
  - Surgery is often necessary to remove infected teeth and treat sinusitis or osteomyelitis. Horses do not shed infected teeth spontaneously.
- g. Malocclusions or abnormalities of the mouth [e.g., brachygnathism (parrot mouth), brachycephaly (sow mouth), sheer mouth, wave mouth, step mouth] produce abnormal wear of the occlusal surfaces of the teeth, interfering with both performance and production. These defects may be breed-related. Many require routine dental attention to promote as much even wear as possible.
- h. Ameloblastoma is the most common neoplasm of dental origin. The tumor, which arises from odontogenic epithelium, is invasive and results in bony degeneration.

## III. DYSPHAGIA

### A. Pharyngitis, laryngitis, and pharyngeal swelling often occur together and concurrently with stomatitis.

- Patient profile and history. Any animal can develop pharyngitis, regardless of age, breed, or gender. There may be a history of traumatic incident or administration of materials via dose syringe, speculum, balling gun, or orogastric intubation. Other historical findings depend on the causative agent.
- Clinical findings. There is often an observable and palpable swelling in the pharyngeal or intermandibular region. There is reluctance or difficulty in eating and swallowing. There may be a mucopurulent nasal discharge and pain often causes the animal to hold the head and neck in extension. The regional lymph nodes may be enlarged and a spontaneous or easily induced cough may be present. The local pharyngeal problem may be accompanied by systemic signs (e.g., fever, tachycardia, tachypnea).
- Etiology and pathophysiology. Pharyngitis may be an extension of stomatitis or, like stomatitis, it may be caused by physical, chemical, or infectious agents.
- Diagnostic plan. A thorough oral examination is necessary to diagnose pharyngitis and determine its causes. Laboratory work, laryngoscopy, endoscopy, radiography, and ultrasonography may all be useful as diagnostic aids.
- Laboratory tests
  - Microbial culture and sensitivity testing may be attempted from a swab of any discharges; however, because the upper alimentary and respiratory tracts are home to a variety of organisms, it may be difficult to interpret the growth of a mixed or contaminated population. Aspiration of abscesses percutaneously through properly prepared sites will yield more reliable results.
  - Fine needle aspiration and cytologic interpretation allows diagnosis of solid-core masses in the pharyngeal region.
  - Hematologic work-up. Hematologic changes may reflect inflammatory changes in the pharynx or a systemic disease.
- Differential diagnoses
  - Rabies (in a domestic animal exhibiting dysphagia, hypersalivation, and mental changes)
  - Respiratory diseases [e.g., strangles, guttural pouch empyema, guttural pouch mycosis, viral respiratory disease, lymphoid follicular hyperplasia (LFH) in horses; calf diphtheria in cattle]

c. Bacterial stomatitis (i.e., oral necrobacillosis, actinobacillosis)

## 7. Specific conditions

### a. Mechanical injury

- (1) Etiology and pathogenesis. Trauma associated with the ingestion of foreign objects or administration of oral medications can lead to bacterial pharyngitis when organisms (e.g., *F. necrophorum*) secondarily infect the site.
- (2) Therapeutic plan. Measures include the following:
  - (a) Penicillin (22,000 IU/kg twice daily intramuscularly)
  - (b) Soft, palatable diets and free-choice water or supportive alimentation through a nasogastric tube (horses)
  - (c) Nonsteroidal anti-inflammatory agents (NSAIDs) or corticosteroids to subdue inflammation and hasten return to normal function
- b. Pharyngeal phlegmon, a sporadic condition affecting adult cattle, produces an acute, deep-seated, diffuse, rapidly spreading cellulitis and inflammation of the oral mucosa and pharynx. The mortality rate is high in acutely affected animals.
  - (1) Etiology and pathogenesis. It is not completely understood what causes pharyngeal phlegmon, but a mixed bacterial population is commonly found in the tissues of the head and neck.
  - (2) Clinical findings include hypersalivation, lacrimation, fever, tachypnea, and tachycardia. There is marked swelling of the tissues of the head. The animal becomes completely anorexic and may die within 24 hours. In more chronic cases, pools of purulent debris build up in the subcutis, imparting a foul odor to the breath.
  - (3) Therapeutic plan. Vigorous treatment, consisting of broad-spectrum antibiotics (e.g., trimethoprim-sulfamethoxazole) and anti-inflammatory agents, is required. Supportive care is also beneficial.

## B. Pharyngeal obstruction

1. Patient profile and history. There may be a history of foreign body ingestion or feeding on solid food objects (e.g., potatoes, turnips, whole cob corn, apples).
2. **Clinical** findings include complete or intermittent dysphagia and the inability to swallow food, water, or both. The animal may make inspiratory noises, and the source of the obstruction may be palpable. Chronic obstruction will result in weight loss, dehydration, or both. Pharyngeal obstruction accompanied by esophageal obstruction will result in bloat in ruminants.
3. Etiology and **pathophysiology**
  - a. Congenital conditions may cause pharyngeal obstruction; however, this is rare.
  - b. Foreign bodies may obstruct the pharyngeal lumen. Examples of foreign bodies include solid feedstuffs like potatoes, apples, or mangos, and nonedible objects, such as rocks, wood, or hard rubber. Pharyngeal obstruction as a result of foreign body ingestion occurs most commonly in mature cattle as a result of their indiscriminate eating habits.
  - c. External masses (e.g., retropharyngeal lymph node adenopathies from infections or tumors) may impinge on the pharynx, displacing normal tissue and causing dysphagia.
    - (1) In cattle, **actinobacillosis** or **lymphosarcoma** can cause external pharyngeal obstruction.
    - (2) In horses, strangles, guttural **pouch disease**, and lymphoid **follicular hyperplasia** can cause dysphagia.
4. Diagnostic plan. A complete **oral** examination is required. Endoscopy is valuable for visualizing the pharynx. Because small, highly portable instruments are available, this procedure can be used in many species under many circumstances.
5. **Laboratory tests.** A leukogram may help confirm the presence of an inflammatory or invasive mass around the pharynx.
6. Differential **diagnoses** include causes of pharyngeal paralysis (e.g., rabies) and other encephalitis or encephalopathies.

## 7. Therapeutic plan

### a. Foreign body obstruction

- (1) When the cause of the obstruction is a foreign body, the object can often be removed manually in a large enough patient. Sedation and the use of a wire loop may be required in some cases.
  - (2) In more complicated cases, surgery followed by antibiotics and supportive care may be required; however, the prognosis is often poor because of the physical trauma sustained.
- b. External masses. The cause of the mass that is impinging on the pharynx must be identified and treated accordingly.

8. Prevention. Providing information to clients about principles of feeding management (e.g., appropriate chopping of tubers, gradual introduction to new feeds) may help minimize the number of foreign body obstructions.

## C. Pharyngeal paralysis

1. Patient profile and history. Subjective and historical information may be of some value for determining the cause of pharyngeal paralysis and dysphagia. For example, the region may be endemic for rabies or the animal may have had access to moldy feed or toxic plants.
2. Clinical findings. In terms of the upper alimentary tract, the clinical findings will not be specific. Findings include dysphagia, ptalism, and, possibly, regurgitation or abnormal vocalization if nerves other than the pharyngeal nerves are paralyzed as well.
3. Etiology and pathophysiology. Pharyngeal paralysis may result from a central or peripheral neurologic disorder; common causes include rabies, botulism, and leukoencephalomalacia.

## D. Esophageal obstruction. In horses and sheep, esophageal obstruction is known as choke. In cattle with complete esophageal obstruction, the most significant clinical finding is the build-up of gas in the rumen; therefore, esophageal obstruction in cattle is discussed in Chapter 3 I B 1.

1. Patient profile and history. Esophageal obstruction may occur in any domestic animal on solid food but is most common in horses, sheep, and cattle.
  - a. Choke is most common in older animals and is often associated with **pelleted** or dry feeds (e.g., beet pulp, oats, bran).
  - b. There may be evidence of insufficient or inaccessible water (e.g., frozen or distant water supplies).
  - c. There may be a history of administration of medicinal boluses.
  - d. Greedy eaten, older animals with defective teeth, and young horses with erupting teeth are susceptible.
  - e. Preexisting esophageal disease, previous episodes of choke, or trauma to the neck are predisposing factors.
2. Clinical findings
  - a. Early findings. Animals will exhibit dysphagia, with extension of the head and neck. Coughing and retching may be accompanied by the discharge of food and frothy saliva from the nostrils. Anxiety, manifested by sweating and **head-shaking**, is common. If the esophageal mass is lodged in the cervical region, it may be palpable percutaneously.
  - b. Late findings include depression and dehydration. The animal may repeatedly attempt to drink without success. Subcutaneous emphysema suggests esophageal rupture. Aspiration pneumonia, manifested clinically as tachypnea, harsh breath sounds, and fever, may occur with high cervical choke.
3. Etiology and pathophysiology. The esophageal diameter may be normal, narrowed, or dilated. Natural areas of esophageal narrowing are the anterior cervical region (in cattle and sheep), the midcervical region (in horses), the thoracic inlet, and the junction of the esophagus and gastric cardia.

- a. External esophageal compression may be brought about by enlargement of normal structures (e.g., thymic **lymphosarcoma**), compressive masses (e.g., abscess), or displacement of structures (e.g., diaphragmatic hernia).
  - b. Luminal obstruction may be the result of ingestion of solid objects (e.g., apples) or dry feed that was inadequately moistened. The latter cause is most common in horses.
  - c. Defects in the esophageal wall (e.g., strictures from healed lesions, ulcers, or congenital or acquired diverticula) can lead to the development of the condition.
  - d. Esophageal spasm has been implicated in some cases.
  - e. Neuropathy. Certain neuropathies (e.g., equine protozoal myelitis, equine herpes virus 1 infection) may interrupt esophageal motility, leading to megaesophagus and esophageal obstruction.
4. Diagnostic plan. The diagnosis is often suspected on the basis of clinical inspection. A nasogastric tube (in horses) or an orogastric tube (in ruminants) can be used to ascertain the site of the obstruction. Endoscopic examination may clarify the diagnosis and offer a prognosis. Radiographic examination using contrast studies allows evaluation of the location and composition of the mass as well as esophageal integrity. (Iodinated organic iodide should be used as a contrast medium if either esophageal perforation or inhalation pneumonia is suspected.) Ultrasonography, fluoroscopy, or both may be warranted in some instances.
  5. Laboratory tests. A hematologic work-up and blood chemistry are important adjuncts to diagnosis and can give some indication of prognosis.
    - a. Packed cell volume (PCV), total serum protein (TSP), and blood urea nitrogen (BUN) evaluations provide information on the animal's hydration status.
    - b. A leukogram should be requested to give an indication of any inflammatory reaction.
    - c. Electrolyte analysis will reveal hyponatremia and hypochloremia in horses, as a result of the loss of large quantities of both sodium and chloride ion in equine saliva. These ion losses and attempts by the kidney to compensate will result in a transient metabolic acidosis followed by a metabolic alkalosis.
  6. Differential diagnoses. The most common differentials for esophageal obstruction are listed in Table 1-3.
  7. Therapeutic plan. This discussion focuses on the treatment of particulate matter (grain) choke in horses.

TABLE 1-3. Differential Diagnoses for Esophageal Obstruction

Neurologic disorders [e.g., rabies and botulism (all species), leukoencephalomalacia and yellow star thistle poisoning (in horses)]
Respiratory diseases in horses [e.g., lymphoid follicular hyperplasia (LFH), strangles, guttural pouch disease, laryngeal scarification following surgery]
Musculoskeletal diseases (e.g., fractured hyoid bone)
Plant toxicoses causing salivation in cattle (e.g., <i>solanum</i> , larkspur, bitterweed)
Mycotoxins causing salivation in cattle (e.g., red clover hay contamination with <i>Rhizoctonia leguminicola</i> )
Metabolic diseases in horses (e.g., eclampsia, hepatic encephalopathy, hypokalemia)
Oral cavity or pharyngeal foreign bodies
Extrapharyngeal masses in cattle (e.g., lymph node abscess, phlegmon)
Anaphylactoid reactions causing pharyngeal swelling
Dental disease
Congenital diseases [e.g., persistent right aortic arch (PRAA), megaesophagus]

- a. Hydraulic flush. All access to grain, hay, edible bedding, and water should be restricted. Warm water is instilled by gravity into the nasogastric tube, causing water and ingesta to flow out of the tube. Often, after a series of hydraulic flushes, the choke will be relieved and the nasogastric tube can be passed into the stomach. Occasionally, the mass can be forced into the stomach by the tube, but this should only be attempted with great care. Xylazine (0.5–1 mg/kg intravenously) is often used in horses to relieve anxiety, provide muscle relaxation, relieve esophageal spasm, and cause lowering of the head to guard against aspiration pneumonia.
  - (1) If the hydraulic flush is not successful, another attempt can be made later. Often, a stubborn choke can be relieved by patience. A horse left without access to feed or water may also resolve the choke spontaneously.
  - (2) If the choke persists for longer than 3 days, a hydraulic flush can be performed with the horse under anesthesia and in lateral recumbency. A cuffed endotracheal tube is placed in the trachea to prevent aspiration of material.
- b. Other measures
  - (1) **Dioctyl sodium sulfosuccinate (DSS)** is a wetting agent that may soften the mass. 10–15 ml of DSS are mixed with water and infused into the nasogastric tube. This treatment is contraindicated if aspiration of material is likely.
  - (2) Suction. A ruminant stomach pump should be used only for suction, not for applying forceful hydraulic pressure against the mass.
  - (3) Atropine (10–30 mg subcutaneously) has been recommended because of its ability to relax smooth muscle, although its efficacy may be limited in horses because of the significant amount of striated muscle in the equine esophagus. Side effects include intestinal colic, ileus, and drying of the mucous membranes.
  - (4) Surgery. Surgical removal of a solid foreign body may be considered but chronic fistulation often develops as a result of poor healing.
- c. Supportive therapy. With a readily resolved choke, supportive therapy is not necessary.
  - (1) Feeding considerations. After the resolution of a long-standing choke, the horse should be held off feed for 2–3 days. Free-choice water should be available, and the horse should be gradually reintroduced to soft feed (mashes) and then a regular diet. Supplementary feeding of grain or pellet gruels via an indwelling nasogastric tube may be necessary in cases of esophageal perforation. Feed placed in an elevated position may help patients with compromised esophageal motility.
  - (2) **Broad-spectrum antibiotics** should be considered if esophageal perforation is suspected or if the choke persists.
  - (3) **NSAIDs** may be administered following resolution of the choke if esophageal irritation is suspected.
  - (4) Intravenous fluids are necessary in cases of chronic choke.
8. Complications. **Sequelae** to choke include esophageal perforation and **cellulitis**, esophageal fistula formation, acute mediastinitis and pleuritis, esophageal stricture, esophageal dilation (**megaesophagus**), recurrent choke, and aspiration pneumonia. All of these complications carry an unfavorable prognosis. The animal should be monitored for 8 weeks after an incident of acute esophageal choke to check for stricture formation.
9. Prevention. Preventive measures include slowing down greedy eaters by feeding small amounts more often, wetting the feed, providing routine dental care, and ensuring that potable water is always available.

### E. Esophagitis

1. Patient profile and history. Esophagitis is more common in companion animals and humans than in the large animal species. If esophagitis is suspected, there may be a history of consumption of irritant chemicals, the chronic retention of an esophageal foreign body, or trauma (e.g., perforation of the esophagus during nasogastric

- intubation). Other historical findings include upper gastrointestinal tract disease or chronic, low-grade abdominal pain.
2. Clinical findings. The signs of esophagitis may be similar to those of choke. Progression of esophagitis may result in an external fistula or extension through fascial planes, producing a cellulitis or thoracic inflammation and a corresponding deterioration of clinical signs.
    - a. Reluctance to eat, anorexia, and dysphagia. Water consumption may or may not be impaired; therefore, hydration status is variable but weight loss is usually evident.
    - b. Pain or anxiety may be apparent on swallowing and there may be pain, swelling, or crepitus over the site of the esophageal lesion.
    - c. Nasal regurgitation. Esophageal spasm may cause the animal to regurgitate food mixed with saliva or blood through the nostrils.
    - d. Signs of aspiration pneumonia (e.g., fever, tachypnea, tachycardia, harsh breath sounds) may be present.
  3. Etiology and **pathophysiology**
    - a. Traumatic ulcerative esophagitis may result when the prolonged presence of a foreign mass in the esophagus causes a pressure necrosis.
    - b. Corrosive esophagitis may result from the ingestion or administration of corrosive materials. Agents that produce stomatitis (e.g., musculoskeletal counterirritants in horses) will also produce esophagitis.
    - c. **Reflux** esophagitis occurs in simple-stomached animals and involves the retrograde movement of gastric or small intestinal fluids (e.g., acid, pepsin, bile salts) into the esophagus. Reflux esophagitis may occur with equine small intestinal obstruction, anterior enteritis, gastric ulcers, or gastric outflow problems.
  4. Diagnostic plan. The diagnostic plan for esophagitis is similar to that for choke. After a thorough oral examination, a nasogastric tube (or orogastric tube in the case of ruminants) should be passed carefully into the stomach. Upon withdrawal, the tube should be examined for the presence of blood, ingesta, and saliva. Endoscopy and double contrast radiography are valuable adjuncts for determining the site and extent of the lesion.
  5. Laboratory tests requested would depend on the etiology of the esophagitis.
  6. Differential diagnoses are similar to those for choke (see Table 1-3).
  7. Therapeutic plan.
    - a. Specific **conditions**
      - (1) Traumatic ulcerative esophagitis. Intensive, conservative therapy may be necessary to remove a foreign mass. High dosages of broad-spectrum antibiotics may be necessary.
      - (2) **Reflux** esophagitis. Gastric distention is treated by decompression via nasogastric intubation. A histamine<sub>2</sub> (H<sub>2</sub>)-receptor antagonist such as cimetidine or ranitidine may be used to decrease gastric acid secretion.
      - (3) Esophageal perforations, strictures, or fistulas may necessitate surgery.
    - b. Supportive therapy. NSAIDs may be helpful. Solid food should be eliminated from the diet until healing has occurred, then solid foods should be introduced gradually. Supplemental feeding via nasogastric intubation may be necessary.
  8. Complications. The **sequelae** to esophagitis may be similar to those of choke.

## STUDY QUESTIONS

DIRECTIONS: Each of the numbered items or incomplete statements in this section is followed by answers or by completions of the statement. Select the **ONE** numbered answer or completion that is **BEST** in each case.

1. Which one of the following statements regarding vesicular stomatitis is true? Vesicular stomatitis:
  - (1) is confined to horses and occasionally swine.
  - (2) is unique in its clinical presentation.
  - (3) is a European disease that is chronic and circulates within herds.
  - (4) produces lesions that are confined to the mucous membranes of the mouth and nasal cavity.
  - (5) is hypothesized to be caused by a mutated plant virus spread by insects.
2. A major difference between foot and mouth disease and swine vesicular disease is that:
  - (1) foot and mouth disease is caused by a **pi**-coronavirus.
  - (2) foot and mouth disease does not affect swine.
  - (3) swine vesicular disease is not reportable.
  - (4) swine vesicular disease virus can persist in processed meats.
  - (5) infection with swine vesicular disease is via the oral route.
3. Which statement regarding bluetongue is true? Bluetongue:
  - (1) causes disease in ruminants and horses worldwide.
  - (2) causes oral disease as well as coronitis and laminitis.
  - (3) is spread by ingestion or aerosol transmission.
  - (4) responds to broad-spectrum antibiotic therapy.
  - (5) causes disease only in animals less than 9 months of age.
4. A set of common dental conditions in domestic animals includes:
  - (1) dentigerous cysts in sheep, periodontal disease, caries.
  - (2) caps, caries, supernumerary molars.
  - (3) sharp edges (or points), supernumerary molars, caps.
  - (4) periodontal disease, dentigerous cysts in sheep, sharp edges (or points).
  - (5) supernumerary incisors, retained incisors, wolf teeth.
5. Which one of the following statements regarding pharyngitis of cattle is **true**?
  - (1) There may be a concurrent mucopurulent nasal discharge.
  - (2) Neurologic disease would not be a differential diagnosis.
  - (3) Bacterial culture will confirm the causative organism.
  - (4) Nonsteroidal anti-inflammatory agents are contraindicated.
  - (5) There is usually little indication of pain.
6. Which one of the following statements regarding pharyngeal **phlegmon** is true? Pharyngeal **phlegmon** is:
  - (1) caused by *Candida* species.
  - (2) a disease of low mortality but high morbidity.
  - (3) a cellulitis of the oral **mucosa** and pharynx.
  - (4) most common in milk-fed calves.
  - (5) responsive to oral astringents.



**DIRECTIONS:** Each of the numbered items or incomplete statements in this section is negatively phrased, as indicated by a capitalized word such as NOT, LEAST, or EXCEPT. Select the ONE numbered answer or completion that is BEST in each case.

7. All of the following statements regarding contagious ecthyma (contagious pustular dermatitis, orf) of sheep are true EXCEPT:

- (1) It is common.
- (2) It is usually diagnosed on the basis of clinical findings.
- (3) It has zoonotic potential.
- (4) It can be eliminated in a flock by the use of systemic antibiotics.
- (5) It produces good immunity in individuals against subsequent infection.

8. Which one of the following statements regarding choke in horses is NOT correct?

- (1) Sedatives are often used in acute equine choke to relieve esophageal spasm.
- (2) Chronic or recurrent choke responds favorably to dietary management.
- (3) Signs resembling dyspnea may predominate early in the course of the condition.
- (4) Central nervous system (CNS) diseases should be included among the differential diagnoses when confronted with a case of possible choke.
- (5) Many grain chokes respond favorably to gentle intraesophageal infusions of water.

By: Dr.Stator = thanatomania 2008  
Egyptian Muslim veterinarian  
dr\_stator\_veto\_18486@yahoo.com

## ANSWERS AND EXPLANATIONS

1. The answer is 5 [I I 4]. Vesicular stomatitis virus affects many species including cattle. The disease appears similar to other vesicular diseases (e.g., foot and mouth disease) and is reportable to federal authorities. This disease is cyclical and seasonal in occurrence but is not enzootic in countries outside of tropical or subtropical climates. Lesions are found on the mucous membranes of the oral and nasal cavity but also on the feet and coronary band.

2. The answer is 1 [I I 5, 6]. Foot and mouth disease affects cloven-hooved animals including swine, and whereas it is caused by a picornavirus, swine vesicular disease is caused by an enterovirus. Swine vesicular disease, like foot and mouth disease, is reportable and can be transmitted through processed meats or other fomites via the oral route.

3. The answer is 2 [I I 8 bl. Bluetongue may cause oral disease, coronitis, laminitis, diarrhea, ocular changes, abortions and congenital defects in offspring. It is a viral disease of ruminants spread by biting flies or semen. Because it is a viral disease, bluetongue does not respond to antibiotics. The disease affects animals of all ages.

4. The answer is 4 [II B C]. Common dental conditions of domestic animals include periodontal disease, sharp edges (or points), supernumerary incisors, dentigerous cysts, wolf teeth, and retained molars or caps. Less common conditions are dental caries, supernumerary molars, and retained incisors.

5. The answer is 1 [III A 2]. A mucopurulent nasal discharge and a cough may accompany cases of pharyngitis because dysphagia may cause pulmonary aspiration of feed. Pharyngitis is often accompanied by palpable swelling in the throat region, and the animal may hold its neck in extension and be reluctant to eat or swallow because of pharyngeal pain. There-

fore, neurological disease (e.g., rabies) should be on the list of differential diagnoses. Bacterial culture does not necessarily confirm the causative organism in the case of bacterial pharyngitis due to the normal, mixed bacterial resident population of the pharynx. Nonsteroidal anti-inflammatory agents may hasten the response as inflammation is subdued.

6. The answer is 3 [III A 7 bl. Pharyngeal phlegmon is a deep-seated cellulitis of the oral cavity, head and pharynx. The cause is poorly understood, although bacteria are believed to be associated with the condition. It affects adult cattle sporadically. Mortality is high because this disease is difficult to treat and poorly responsive to any conventional therapy.

7. The answer is 5 [I I 9]. Contagious ecthyma (contagious pustular dermatitis, orf) cannot be eliminated in a flock of sheep by using systemic antibiotics. Antibiotics have no efficacy against the causative agent, a parapoxvirus that maintains persistence within a flock. Contagious ecthyma is a common disease that is usually diagnosed on the basis of clinical findings. It has zoonotic potential. Infection produces good immunity in animals that have been exposed to the virus.

8. The answer is 2 [III D]. Chronic or recurrent esophageal obstruction (choke) is likely to be attributable to internal or external esophageal stricture; therefore, dietary management (such as the feeding of slurries) is usually ineffective in the long term. Sedatives are often used in equine patients to relieve esophageal spasm in patients with acute choke. Signs resembling dyspnea may predominate early in the condition. Central nervous system (CNS) disorders must be ruled out when making the diagnosis. Many grain chokes respond favorably to hydraulic flush.